AMENDMENTS TO THE SPECIFICATION

Please replace the paragraph bridging pages 2 and 3 with the following amended paragraph:

Meanwhile, chronic renal failure is a disease with characteristics of glomerulosclerosis and renal interstitial fibrosis. It is believed that the qualitative change and/or quantitative increase of extracellular matrix components is the main mechanism of the onset and progress of the disease. It has been known that transforming growth factor β (TGF-β) is a differentiation and growth factor with a wide range of various physiological functions. It has also been known that transforming growth factor β (TGF-β) has an activity responsible for the qualitative change and quantitative increase of components of extracellular matrix (Border W. A., *et al.*, *N. Engl. J. Med.*, **331**, 1286-1292, 1996; Roberts, A. B., *et al.*, *Proc. Natl. Acad. Sci. U.S.A.*, **83**, 4167-4171, 1986; Fukabori, Y., *et al.*, *Int. J. Urol.*, **4**, 597-602, 1997; Lohr, M., *et al.*, *Cancer Res.*, **61**, 550-555, 2001). Since it was shown that in an experiment using a renal failure model, the introduction of the gene of decorin, a protein suppressing the specific action of TGF-β (Isaka Y., *et al.*, *Nature Med.*, **2**, 418-423, 1996) and the administration of anti-TGF-β antibodies (Ziyadeh F.N., *et al.*, *Proc. Natl. Acad. Sci. U.S.A.*, **97**, 8015-8020, 2000; Sharma K., *et al.*, *Diabetes*, **45**, 522-530, 1996; and Border W.A. *et al.*, Nature, **346**, 371-374, 1990) were effective, it is considered that the suppression or inhibition of the physiological functions of TGF-β can lead to the treatment of chronic renal failure.

Please replace the paragraph bridging pages 38 and 39 with the following amended paragraph:

According to a laboratory manual [Masato Okada and Kaoru Miyazaki, "Kaitei, *Tanpakushitsu Jikken Noto*, Jyo (*Notebook for Protein Experiments*, Vol.1, revised edition)", Yodo-sha, p.162-79], a fusion protein (GST-MDTS9A) of a peptide consisting of the 280th to 410th amino acids in the amino acid sequence of SEQ ID NO:2 and glutathione S-transferase (GST) was produced in an inclusion body fraction, using a pGEX-6P-1 plasmid (manufactured by Amersham Pharmacia Biotech) as an expression vector and *E. coli* as a host cell. A preparative SDS-polyacrylamide gel electrophoresis (PAGE) was carried out on

the inclusion body fraction, to extract the intended GST-MDTS9A protein from the gel by a diffusion method [Masato Okada and Kaoru Miyazaki, "Kaitei, *Tanpakushitsu Jikken Noto*, Ge (*Notebook for Protein Experiments*, Vol.2, revised edition)", Yodo-sha, p.48-51].

Please replace the paragraph bridging pages 44 and 45 with the following amended paragraph:

The pGV-B2-MDTS9pro5k obtained in Example 1 was introduced in the HEK293-EBNA cell by the method described in Reference Example 3, and cultured for 16 hours, followed by washing twice with PBS. Subsequently, the culture medium was replaced with DMEM (GIBCO-BRL), 100 μg/mL penicillin, 100 μg/mL streptomycin, and 250 μg/mL G418 (manufactured by Nakarai Tesque, Inc.), for culturing for 6 hours (serum-free culture hereinbelow). Continuously, then, TGF-β (manufactured by Sigma) or IL-1 (manufactured by Sigma) was added and the luciferase activity was assayed after culturing for 24 hours. As in Example 2, the assay value was corrected on the basis of the activity value of β-gal. Consequently, an about 1.7-fold increase of the luciferase activity was observed by the addition of TGF-β (1 ng/ml), while an about 0.7-fold or less decrease of the luciferase activity was observed, by the addition of IL-1 (1 ng/ml) (Figs. 1 and 2). This indicates that at least a TGF-β response region and at least an IL-1 response region exist in the DNA fragment. Meanwhile, the luciferase activity in the cell in which pGV-B2-MDTS9pro2k was introduced was also assayed by adding TGF-β to the cell. The resulting luciferase activity was at the same level as in the control.